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Subcorneal pustular dermatosis associated with *Mycoplasma pneumoniae* infection

To the Editor: Subcorneal pustular dermatosis (SPD) or Sneddon-Wilkinson disease is a rare, chronic, and relapsing skin disorder, consisting of a symmetric sterile pustular eruption that primarily involves trunk and large skin folds. Flaccid, subcorneal pustules are half-filled with pus with a hypopyon-like appearance, containing polymorphonuclear neutrophils. The pathophysiology is unknown, but common associations include monoclonal IgA paraproteins, inflammatory bowel disease, and rheumatoid arthritis. We describe a rare association of SPD with *Mycoplasma pneumoniae* infection.

A 36-year-old woman, without significant personal or familial medical history (including psoriasis), presented with a rash for the last 5 days. She reported a dry cough without fever, night sweats, or other symptoms for the last week. Cutaneous examination revealed a pustular eruption on the trunk, proximal limbs, and large folds (Fig 1, *A*), consisting of large pustules with hypopyon-like accumulation of pus (Fig 1, *B*). Face, scalp, palms, soles, nails, and mucous membranes were spared. The rest of physical examination revealed unremarkable findings. No drug had been introduced before the eruption. Histologic examination showed a subcorneal unilocular pustule without spongiosis containing polymorphonuclear neutrophils with rare acantholytic keratinocytes. Direct immunofluorescence did not show immunoglobulin or complement deposition. Bacteriologic, virologic, and mycologic cultures of pus produced negative results, as did indirect immunofluorescence. Laboratory tests found mildly elevated C-reactive protein. These findings were consistent with the diagnosis of SPD.

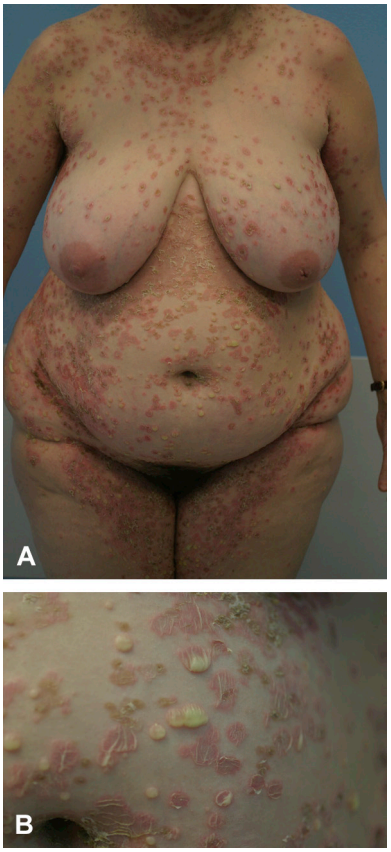

Fig 1. Subcorneal pustular dermatosis. **A**, Pustular eruption on the trunk, the proximal limbs, and in the large folds. **B**, Hypopyon-like accumulation of pus.

Antinuclear antibodies, serum protein electrophoresis, plasma immunoglobulin levels, and circulating lymphocyte flow cytometry produced normal or negative findings. *M pneumoniae* IgM and IgG antibodies were elevated, suggesting recent infection. Throat swab polymerase chain reaction was positive for *M pneumoniae*. Chest x-ray and thoracic-abdominal-pelvic computed tomography scan revealed unremarkable results. Oral dapsone was started but stopped by the patient in a few days because of nausea. Cutaneous lesions disappeared in a few weeks with topical corticosteroids alone. No recurrence was noted at 18 months' follow-up.

In spite of rapidly favorable outcome without recurrence, the clinical and histologic findings were consistent with SPD. We believe the acute course of the disease may suggest an infectious origin. In our case, the patient did not have pneumonia but a mild upper respiratory tract infection that could have gone unnoticed. Skin lesions responded to topical corticosteroids alone, suggesting that systemic treatment may not be necessary.

Two similar cases of subcorneal pustules after *M pneumoniae* respiratory infections have been

described, by Papini et al[1] in an 8-year-old boy and by Winnock et al[2] in a 43-year-old man. In these 2 cases, dapsone was prescribed without recurrence. Interestingly, *M pneumoniae*—induced Stevens-Johnson syndrome with subcorneal pustules has been described a few times.[3-5] However, in our observation mucous membranes were spared, rendering Stevens-Johnson less plausible. We suggest that there may be a specific presentation of SPD of infectious origin, possibly more frequent in young patients. Our observation highlights the importance of looking for *M pneumoniae* in cases of SPD, even without respiratory symptoms, and suggests topical treatment for a few weeks may be sufficient if the eruption is considered secondary to *M pneumoniae* infection.

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Rapid deterioration in a patient with primary aggressive cutaneous epidermotropic CD8+ cytotoxic T-cell ('Berti') lymphoma after administration of adalimumab

To the Editor: Primary aggressive cutaneous epidermotropic CD8+ cytotoxic T-cell lymphoma, a World Health Organization provisional entity here referred to as "Berti lymphoma," comprises less than 1% of cutaneous T-cell lymphomas (CTCLs).[1] Berti lymphoma classically presents as eruptive hyperkeratotic plaques or nodules with a phase of ulceration and necrosis.[2] The epidermis exhibits epidermotropism of atypical pleomorphic lymphocytes and occasional necrotic keratinocytes.[2] Dissemination to lung, testis, central nervous system, and oral mucosa with sparing of lymph nodes is common.[3] We present a case of Berti lymphoma that arose following a trial of adalimumab in the setting of a long-standing spongiotic and psoriasiform dermatitis clinically suggestive of psoriasis.

A 35-year-old African American woman had a 10-year history of nonspecific "dermatitis." In the year before her death, she was admitted to her local hospital several times for shortness of breath and dermatitis and was repeatedly treated with prednisone. Skin biopsies from this period showed a nonspecific spongiotic and psoriasiform dermatitis. Immunohistochemical analysis failed to demonstrate any concerning atypical or CD8+ epidermotropic infiltrate. She was seen by a local dermatologist as an outpatient, psoriasis was diagnosed, and a single loading dose of adalimumab (80 mg) was administered. She presented 2 days later to her local hospital for respiratory decline and rapidly developing cutaneous ulceration.

On transfer to our institution's intensive care unit 6 days later, examination revealed skin ulcers localized primarily to the trunk as well as serpiginous to arcuate scaly plaques and erosions on the extremities (Fig 1). A biopsy on an ulcer edge revealed an epidermotropic infiltrate of atypical CD8+ cells consistent with a diagnosis of Berti lymphoma (Fig 2). Atypical cells stained diffusely positive by immunohistochemistry for CD3, CD8, granzyme B, and TIA-1, with 70% of cells proliferating by Ki-67. A subpopulation of atypical cells were CD56 positive. Atypical cells were negative for CD4, CD5, CD7, CD20, CD30, CD57, CD99, HSV, and TdT. Flow cytometry of peripheral blood was normal, and HTLV I/II and EBV were negative. Left upper and lower lung biopsies revealed focal involvement by CD8+ and CD56+ atypical large lymphocytes consistent with pulmonary lymphoma. Respiratory function deteriorated, and she died 2 months after admission.

Adalimumab, a human tumor necrosis factor (TNF)-alpha monoclonal antibody, is indicated to treat a range of autoimmune diseases including psoriasis.[4] Whether TNF-alpha blockade is associated with development of lymphomas is controversial, but rapid and fatal progression of